Prevention of Trichobezoar in the Cotton Rat, *Sigmodon hispidus hispidus*[1]

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Until 1939 the cotton rat, *Sigmodon hispidus hispidus*, was not commonly employed in experimental work. However, since the studies of Armstrong (*1*), many investigators have used this rodent in biological studies (*3, 5–9*). In spite of its increasing popularity in experimental work, a review of the literature reveals no mention of trichobezoar formation in this animal.

In our studies of experimental caries in the cotton rat (*4*) the mortality rate was found to be about 40% during a 14-week experimental period. Autopsy of those animals dying prior to the termination of the experiment consistently revealed trichobezoar formation. It occurred to one of us (C. M. McC.) that the addition of cellulose to the diet might prevent this condition.

One group of 51 weanling cotton rats (Group I) received the basic sugar caries-producing synthetic diet of Shaw, *et al.* (*8*), and a second group of 15 weanling cotton rats (Group II) received the same diet with the addition of 10% by weight of finely divided cellulose. The latter was so distributed throughout the diet that it was impossible for the rats to separate and reject it.

Group I was divided into three equal subgroups in such a manner that a rat in subgroup A had a litter mate in subgroups B and C. Group II was similarly divided.

TABLE 1

Comparison of Weights Gained and Incidence of Trichobezoars Between Group I, Receiving No Cellulose, and Group II, Receiving 10% Cellulose in the Diet*

	Group I—Caries diet					Group II—Caries diet + 10% cellulose				
	Total No. of rats	Total No. of litters	Total wt. gained (gm)	Mean wt. gained (gm)	Incidence of tricho-bezoars	Total No. of rats	Total No. of litters	Total wt. gained (gm)	Mean wt. gained (gm)	Incidence of tricho-bezoars
Subgroup A—all rats; distilled drinking water only	17	12	639	37.6	17 +	5	4	412	82.4	5 –
Subgroup B—all rats; Group I = .02% potassium oxalate in distilled drinking water Group II = .0002% potassium oxalate in distilled drinking water	17	12	734	43.2	17 +	5	4	395	79.0	5 –
Subgroup C—all rats; Group I = .22% potassium oxalate in distilled drinking water Group II = .002% potassium oxalate in distilled drinking water	17	12	743	43.7	17 +	5	4	396	79.2	5 –
Subgroup A—only those rats living 14 weeks; distilled drinking water	9	9	367	40.8	9 +	5	4	412	82.4	5 –

t value at 1% level = 3.55—$P < .01$ ∴ difference is significant.

*Difference in weights gained of rats surviving 14 weeks in subgroups IA and IIA was tested for significance.

One-third of Group I and Group II (control subgroups IA and IIA) drank distilled water, while to the drinking water of the remaining two-thirds (subgroups IB,IC and IIB,IIC) varying amounts of potassium oxalate were added. This regime was maintained for a period of 14 weeks.

Whereas the above method of procedure was originally designed to determine the role of oxalates in rat dental caries, the same animals were used concurrently to observe the effects of cellulose on trichobezoar formation.

The environmental conditions of both groups were identical, and the temperature and humidity of the animal laboratory were kept at a constant level at all times.

Rats dying during the course of the experiment were weighed and autopsied and the data recorded. With the termination of the experimental period all surviving rats

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were killed and were similarly weighed, autopsied, and examined for trichobezoar.

Of the rats in Group I receiving the basic sugar caries-producing synthetic diet, a total of 21 died before the end of the 14-week period, giving a mortality rate of about 40%. Postmortem examination of all Group I rodents consistently showed trichobezoar formation. The size of the trichobezoars varied somewhat, but in those rats that died during the experiment they were apparently of sufficient size to interfere effectively with gastric emptying. These trichobezoars were at least as large as one-half of the stomach volume, and some were so large as to fill the stomach completely. Multiple trichobezoars were found in several of the rats, but no more than three were found in any one animal. All other organs appeared normal on gross examination.

Of the Group II animals receiving the basic sugar caries-producing diet with the addition of 10% cellulose, all lived the full 14 weeks and appeared quite normal in contrast to the moribund appearance of the previous group. Autopsy of these rats showed no evidence of trichobezoar formation, and all organs and tissues were grossly normal.

Comparison of the weight gains of the rats (Table 1) indicates the marked difference between the two major groups. This difference was shown to be significant by comparison of subgroups IA and IIA according to accepted statistical methods (*2*). Since an analysis of the weight gains of the rats showed no significant difference between A and B, A and C, and B and C within both major groups, it was felt that Groups I and II, as a whole, could logically be compared for weight gains by analysis of the control subgroups IA and IIA. As seen in Table 1, only those rats of Group I which survived the full 14 weeks were used for evaluation. The t value between the control subgroups demonstrates that there is a significant difference in the weights gained, indicating that the cellulose may have been the responsible factor.

The possible effects of the oxalates on trichobezoar formation may be discounted by comparison of trichobezoar distribution among the subgroups of major groups I and II (Table 1). Since the trichobezoars were distributed equally throughout the control rats and rats receiving oxalates of Group I (100% trichobezoars), and none was found in the three subgroups of Group II, it may be assumed that the oxalates played no role in trichobezoar formation.

The consistent appearance of trichobezoars in rats on a synthetic diet without cellulose, compared with its absence in those animals on a synthetic diet with cellulose, seems to point fairly conclusively to the advantage of this type of inert material in the diet.

References

1. Armstrong, C. *Publ. Hlth Rep.*, 1939, **54**, 1719–1721.
2. Croxton, F. E., and Cowden D. J. *Applied general statistics.* New York: Prentice-Hall, 1945. Pp. 322, 324.
3. Eaton, M. D., Meikeljohn, G., and Van Herick, W. *J. exp. Med.*, 1944, **79**, 649–669.
4. Howell, S. R., Schlack, C. A., Taylor, B. L., and Berzinskas, V. J. Project X–418, Rep. No. 7, Naval Medical Research Institute, November 6, 1947.
5. Jungblut, C. W. *Proc. Soc. exp. Biol. Med.*, 1943, **43**, 479–486.
6. Meyer, B. J., and Meyer, R. K. *J. Mammal.*, 1944, **25**, 107–129.
7. Schweigert, B. S., Potts, E., Shaw, J. H., Zepplin, M., and Phillips, P. H. *J. Nutrition*, 1946, **32**, 405–412.
8. Shaw, J. H., Schweigert, B. S., McIntire, J. M., Elvehjem, C. A., and Phillips, P. H. *J. Nutrition*, 1945, **28**, 333–345.
9. Steinbach, M. M., and Duncan, C. J. *Proc. Soc. exp. Biol. Med.*, 1944, **44**, 288–290.

An Experimental Syndrome of Fatty Liver, Uric Acid Kidney Stones, and Acute Pancreatic Necrosis Produced in Dogs by Exclusive Feeding of Bacon

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In the experiments to be described,[1] 9 dogs were fed on fat bacon exclusively. Two of these died after 2½ and 3 months, respectively. On postmortem examination they showed a severe fatty degeneration of the liver without significant changes in other organs.

Five dogs lived for periods varying between 5 and 7 months after the bacon diet was begun. At first these animals did well, but soon their appetite declined; they vomited occasionally and had to be fed forcefully. Occasionally they suffered from diarrhea. There was severe weight loss, and a peculiar kind of skin sores developed on the front and hind legs. Although the general condition became very poor and remained so for weeks, death occurred suddenly and often unexpectedly.

The postmortem examination of these animals revealed characteristic findings. The liver showed extensive fatty infiltration. In most cases only a few normal liver cells remained. These were located at the periphery of the lobules. Four of the 5 dogs had stones in the pelvis of the kidneys, in the ureters, or in the bladder which varied in size from a pinhead to a cornseed. They were thin and flat, sometimes round or polygonal in shape, and were of a greenish-yellow or brownish color. The bladder showed an apposition of stony material on the mucosa. The stones burned completely on heating and dissolved in alkali. Dried and powdered, the material gave a strong murexide reaction. Eighty-five per cent of the dry weight of the stones could be accounted for as uric acid. When the stones were dissolved in dilute ammonia, the solution filtered, and hydrochloric acid added, typical crystals of uric acid appeared.

In addition to these findings, 4 of the 5 dogs showed macroscopic and microscopic severe changes in the pancreas. The first animal showed extensive necrosis of the

[1] These experiments were carried out in the period 1938–40. With the German invasion of the Netherlands and the ensuing shortage of food, they had to be discontinued, and publication of the results was consequently delayed.